Journal of Cardiology 69 (2017) 389–393

Contents lists available at ScienceDirect

Journal of Cardiology

journal homepage: www.elsevier.com/locate/jjcc

Original article

Estrogen variation during the menstrual cycle does not influence left ventricular diastolic function and untwisting rate in premenopausal women

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ARTICLE INFO

Article history:
Received 15 July 2016
Received in revised form 6 September 2016
Accepted 16 September 2016
Available online 22 October 2016

Keywords:
Diastolic function
Premenopausal woman
Menstrual cycle
Estrogen
Echocardiography

ABSTRACT

Background: Although a cardioprotective effect of estrogen has been suggested by experimental studies, clinical data on the influence of estrogen on left ventricular (LV) diastolic function are sparse. The LV untwisting rate obtained by 2D speckle tracking echocardiography (2D-STE) is correlated with the time constant of LV pressure decay (tau), and this correlation is independent of left atrial pressure. Therefore, we used conventional Doppler echocardiography and 2D-STE to investigate changes in LV diastolic function during a single menstrual cycle in premenopausal women.

Methods: Twenty healthy premenopausal woman (mean age, 28.1 ± 2.7 years) were enrolled. Clinical and echocardiographic data were obtained during the follicular phase (F-phase) and luteal phase (L-phase) of a single menstrual cycle. We compared the clinical and echocardiographic data, and estrogen levels between the two phases.

Results: There were no significant differences in LV diastolic parameters derived from Doppler echocardiography (E/A, $p = 0.295$; E/e', $p = 0.449$, DcT, $p = 0.178$) or 2D-STE (peak untwisting rate, $p = 0.892$; time-to-peak untwisting, $p = 0.951$) between the two phases of the menstrual cycle. However, there was a significant decrease in estrogen levels between the F- and L-phases (177 ± 119 pg/ml vs. 35 ± 12 pg/ml, $p < 0.0001$).

Conclusions: LV diastolic function in healthy premenopausal women did not significantly change during the menstrual cycle. Estrogen does not appear to have a significant acute effect on LV diastolic function in premenopausal woman.

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Introduction

The prevalence of diastolic heart failure (HF) is increasing with the advancing age of society, and the prevention and treatment of diastolic HF has become an important health concern [1,2]. Diastolic HF is more prevalent in women than in men, especially postmenopausal women [2–4]. Some previous investigations [5,6] showed a sharp decrease in left ventricular (LV) diastolic function in healthy women after 50–60 years of age compared with men, suggesting that the loss of a cardioprotective effect of estrogen after menopause may accelerate the progression of LV diastolic dysfunction with advancing age in women. Indeed, some animal studies [7–11] have shown that estrogen has a cardioprotective effect, and its deficiency may result in a deterioration of LV diastolic function. However, there is a paucity of clinical data regarding the effect of estrogen on LV diastolic function, and the effect of estrogen on LV diastolic function in the clinical setting is still controversial. Serum estrogen levels show a dynamic change throughout the menstrual cycle in premenopausal women. Some investigators showed that LV diastolic function was improved in

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http://dx.doi.org/10.1016/j.jjcc.2016.09.008
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the luteal phase [12,13], whereas others found no significant fluctuation in LV diastolic function during the menstrual cycle [14]. Noninvasive echocardiography was used to assess LV diastolic function in these previous studies. However, the conventional diastolic parameters are known to have their inherent limitations [15].

Advancements in 2D speckle tracking echocardiography (2D-STE) have provided more detailed insight into cardiac function than conventional echocardiography. The LV untwisting rate using 2D-STE was reported to be correlated with the time constant of LV pressure decay (tau), the gold standard for LV diastolic function, and the correlation was independent of left atrial (LA) pressure [16–18]. We hypothesized that assessment of LV diastolic function using 2D-STE might reveal the effect of estrogen on LV diastolic function more clearly than the conventional diastolic parameters assessed in previous investigations.

The purpose of this study was to investigate the influence of estrogen on LV diastolic function, using conventional echocardiography and 2D-STE in premenopausal woman during the menstrual cycle.

Materials and methods

Study population

Twenty healthy premenopausal women (24–35 years; mean, 28.1 ± 2.7 years) without a history of pregnancy were enrolled in this study. Their menstrual cycles were regular for more than 3 months before the study, and none of them was taking oral drugs including contraceptive pills at the time of enrollment. All participants were asymptomatic, normotensive, nondiabetic, nonsmokers, and had no significant medical history. Written informed consent was obtained before the study from all participants.

Study design

Each participant was studied twice during one menstrual cycle. The measurement was done in the follicular phase (F-phase) and the luteal phase (L-phase). The F-phase was a period from 2 to 4 days after the beginning of menstruation, whereas the L-phase was a period from 7 to 10 days before the predicted beginning of the next menstruation. In each phase, blood sampling and an ultrasound examination were performed on the same day.

Clinical and laboratory parameters

Clinical parameters including systolic blood pressure, diastolic blood pressure, and heart rate were measured in each participant. Blood sampling was performed on the morning of the ultrasound examination to measure complete blood count, and serum concentrations of estradiol and progesterone. Serum estradiol and progesterone levels were measured by an electrochemiluminescence immunoassay (ECLIA). Serum was obtained by centrifugation at 1000 × *g* for 10 min and kept at −80 °C before assay.

Conventional 2D and Doppler echocardiography

A standard two-dimensional and Doppler echocardiographic examination was performed using a Vivid 7 ultrasound machine (GE Medical Systems, Milwaukee, WI, USA) with an M4S probe. Our echo laboratory is maintained under the guidelines of the Japanese Society of Echocardiography [19]. In all subjects, LV and LA chamber quantification were performed according to guidelines of the American Society of Echocardiography (ASE) [20]. LV end-diastolic and end-systolic volumes were determined in the apical 2- and 4-chamber views using modified biplane Simpson's method, and these volumes were used to calculate LV ejection fraction (LVEF). Maximum LA volume was measured using the biplane area-length method and was indexed by body surface area (LA volume index).

To assess LV diastolic function, pulsed-wave Doppler imaging of mitral inflow and tissue Doppler imaging (TDI) of mitral annular motion at the septum were performed as recommended by the ASE [15]. The peak velocity of early (E) and late (A) diastolic filling mitral flow, and the early flow deceleration time (DcT) were measured using pulsed wave Doppler (the *E*/*A* ratio was calculated). Early diastolic annular velocity (*e*′) was measured from TDI of mitral annular motion at the septum in the apical 4-chamber view, and the ratio of peak early diastolic mitral inflow velocity to annular velocity (*E*/*e*′) was calculated [15].

We scanned apical and basal short-axis planes using a high-frame rate (70 ± 20 frames/s) with second harmonic B-mode for 2D-STE. We defined the proper short-axis levels as follows [16,21]: the basal level, the mitral valve, the apical level, and the LV cavity alone with no visible papillary muscles. The LV cross section was made as circular as possible. LV twisting and untwisting analysis were performed offline on a PC workstation using commercially available software (EchoPAC, GE Medical Systems).

Terminology and calculation of LV twisting and untwisting

The LV performs a wringing motion with a counterclockwise rotation at the apex and a clockwise rotation at the base [16,21], and LV twisting (degrees) is defined as apical rotation relative to the base. Counterclockwise LV twisting as viewed from the apex is expressed as a positive value. The opposite rotation after LV twisting is defined as the LV untwisting (degrees). We used the time derivative of untwisting, (untwisting rate, degrees/s) and the time from QRS onset to peak untwisting rate (time-to-peak untwisting) as surrogate markers of LV diastolic function [16,21] (Fig. 1).

For temporal analysis, the time sequence was normalized to the percentage of systolic duration, with the beginning and end of systole defined as the QRS onset and aortic valve closure, respectively. The time-to-peak untwisting was expressed as a percent of the systolic duration (% systolic duration), as previously described [21].

Statistical analysis

All statistical analyses were performed using SPSS 17.0 (SPSS Inc., Chicago, IL, USA). All variables were expressed as mean ± standard deviation. All values obtained during F- and L-phases were compared by a paired *t*-test. For all analysis, a probability value <0.05 was considered statistically significant.

Results

The results are shown in Tables 1 and 2. Comprehensive data were obtained from all participants.

Table 1
Patient characteristics.

	F-phase	L-phase	*P* value
SBP (mmHg)	101 ± 9	105 ± 10	0.158
DBP (mmHg)	63 ± 9	62 ± 7	0.784
Heart rate (beats/min)	67 ± 9	67 ± 5	0.830
Hemoglobin (g/dl)	12.8 ± 1.1	13.0 ± 1.0	0.126
Hematocrit (%)	38.5 ± 3.4	39.4 ± 2.6	0.107
Estrogen (pg/ml)	35 ± 12	177 ± 119	0.005
Progesterone (ng/ml)	0.6 ± 0.2	6.6 ± 8.3	0.005

F-phase, follicular phase; L-phase, luteal phase; SBP, systolic blood pressure; DBP, diastolic blood pressure.

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